Exhibit G

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UNITED STATES DISTRICT COURT

NORTHERN DISTRICT OF CALIFORNIA

SAN FRANCISCO DIVISION

IN RE: VOLKSWAGEN "CLEAN DIESEL" MARKETING, SALES PRACTICES, AND PRODUCTS LIABILITY LITIGATION

CASE NO. 3:15-md-02672-CRB

Hon. Charles R. Breyer

THIS DOCUMENT RELATES TO:

All Cases

EXPERT REPORT OF DR. GEORGE D. THURSTON, SC.D.

Attached as Exhibit 1 is the expert report of Dr. George D. Thurston, SC.D.

EXHIBIT 1

EXPERT REPORT OF

OF

DR. GEORGE D. THURSTON, SC.D.

RE: VOLKSWAGEN GROUP OF AMERICA, INC. ET AL,

CASE NO. 2672 CRB (JSC)

1. BRIEF EXECUTIVE SUMMARY

In September 2015, the U.S. Environmental Protection Agency ("EPA") and the California Air Resources Board ("CARB") issued a notices of violation to Volkswagen AG, Audi AG, Volkswagen Group of America, Inc., (collectively "Volkswagen") Porsche AG, and Porsche Cars North America, Inc., alleging that certain Volkswagen turbocharged direct injection ("TDI") diesel vehicles in the United States were equipped with "defeat device" software ("Defeat Device").[1] Volkswagen's Defeat Device on TDI vehicles detects whether a vehicle is being tested, and, if it is, controls the engine performance during testing so that emissions temporarily operate within legal limits.[2] According to the State of California, Volkswagen has admitted that, since at least 2008, such Defeat Devices have been used to evade California and federal emissions standards for its supposedly "clean" diesel vehicles.[3] Under real-world driving conditions on the road, however, the vehicles emit nitrogen oxides ("NOx") into the air at levels more than 30 times and up to 35 times the legal limits provided by California and federal law.[4]

According to the State of California, that state's consumers make up the largest state auto market in the United States, and a significant portion of the affected vehicles were sold in California to California consumers.[5] Per the State of California, Volkswagen and Porsche have introduced, or caused to be introduced, approximately 87,000 non-compliant 2.0 and 3.0 liter vehicles into California, which in turn have emitted thousands tons of excess NOx and related toxic pollutants year after year.[6] In addition, per the Second Partial Consent Decree,

[1] 3.0-Liter Consumer Class Action Settelment Agreement And Release (Amended).
[2] California's Civil Enforcement Complaint at ¶ 1.
[3] *Id.*
[4] Volkswagen AG Rule 11 Plea Agreement at ¶ 34.
[5] California's Civil Enforcement Complaint at ¶ 6.
[6] *Id*. at ¶ 2.

approximately 580,000 subject vehicles (approximately 500,000 of the 2.0 Liter TDI vehicles and approximately 80,000 of the 3.0 Liter TDI vehicles) were introduced nationwide.[7]

By selling these cars with Defeat Devices, Volkswagen caused a significant amount of excess NOx tailpipe emissions. These excess emissions directly resulted in increased human exposures to NOx air pollution in the ambient air, in the form of nitrogen dioxide (NO_2), which has known adverse human health effects when breathed. In addition, these primary NOx emissions are known to react in the atmosphere to secondarily form fine particulate matter ($PM_{2.5}$) and ozone (O_3) air pollution, both of which also have their own known adverse human health effects. I therefore conclude that these excess NOx emissions, because of their associated increments in population air pollution exposures to NO_2, O_3, and $PM_{2.5}$, coming in addition to prevailing levels of ambient air pollution, have therefore caused increases in adverse health events that would not have occurred without these excess emissions.

2. INTRODUCTION

A. Education/Qualifications:

I am George D. Thurston, Sc.D. I am a Professor at the New York University School of Medicine in the Department of Environmental Medicine, where I am the Director of the Program in Human Exposures and Health Effects of Air Pollution. I received my undergraduate degree in Engineering from Brown University, and my Doctorate of Science (Sc.D.) in Environmental Health Sciences from the Harvard University School of Public Health. I have published extensively regarding the human health effects of inhaled air pollutants, particularly in relation to asthma attacks, hospital admissions, and human mortality. I have been called upon by both the

[7] Second Partial Consent Decree.

U.S. House of Representatives and the U.S. Senate multiple times to provide testimony before them regarding the human health effects of air pollution.

I have served as an advisor to the U.S. EPA regarding the human health effects of air pollution as a member of the Clean Air Scientific Advisory Committee (CASAC) panel on Sulfur Oxides and Nitrogen Oxides, and as a contributing author of various EPA Integrated Science Assessments (ISAs), which are relied upon by the EPA to set air pollution air quality standards in the US.

My business address is: Three Catherine Ct., Chester, NY 10918. The publications reviewed or relied upon for this testimony are listed at the end of this report as "Literature Cited," as well as a list of my publications in the last 10 years and a list of cases in which I have testified as an expert in the last 4 years. I offer this report on behalf Plaintiffs in the Consumer Opt-Out Cases in the Volkswagen MDL, No. 2672 (Breyer, J.).

B. Compensation:

I am being compensated at a rate of $300/hr. for time spent on report development and other related time spent on this case, and $600/hr. for time spent giving testimony in this case, should that occur.

C. Expert Assignments In This Case:

For this report, I was asked to review documents related to this case to gain an understanding of excess Volkswagen TDI vehicle emissions resulting from use of the Defeat Devices, and to draw conclusions on the impact to human health, based on my knowledge of the subject of air pollution and its human health effects, especially regarding the human health implications of those excess air pollution emissions into the air.

D. Brief Summary of Work Performed:

The purpose of this report is to document the adverse human health effects that are associated with exposures to air pollutants emitted from those Volkswagen TDI vehicles that emitted many times their EPA designated legal emission limits for nitrogen oxides (NOx). For this work, I reviewed the documents provided, and prepared this report regarding the human health implications of the air pollution emissions caused by Volkswagen's use of Defeat Devices. I consider the adverse health impacts that occurred as a result, both from the directly emitted NOx, as well as from the secondary fine particulate matter ($PM_{2.5}$) and ozone (O_3) that resulted in the atmosphere from the original NOx emissions. The United States 1970 Clean Air Act and its amendments require the US Environmental Protection Agency (EPA) to set National Ambient Air Quality Standards (NAAQS) for six common air pollutants (also known as "criteria air pollutants") in outdoor air with the aim of reducing the health risks associated with those pollutants in our air. Fossil fuel powered vehicles, such as those considered here, are major sources of emissions of criteria air pollutants or their precursors, including fine particulate matter ($PM_{2.5}$), nitrogen dioxide (NO_2), and ground-level ozone (O_3). My testimony focuses in particular on the health impacts from these three pollutants (nitrogen dioxide, ozone, and fine particulate matter).

3. SUMMARY OF OPINIONS:

a. Increased NOx emissions from VW TDI automobiles have resulted in increased adverse human health impacts.

b. Increased NOx emissions from VW TDI automobiles have resulted in increased ozone air pollution, which has resulted in additional adverse human health impacts.

c. Increased NOx emissions from VW TDI automobiles have resulted in increased particulate matter air pollution, which has resulted in added human health impacts.

4. BASES FOR MY OPINIONS:

A. Increased NOx Emissions From VW TDI Automobiles Have Resulted In Increased Adverse Human Health Impacts.

The adverse human health consequences of breathing air pollution are well documented in the published medical and scientific literature. During recent decades, medical research examining air pollution and public health has definitively shown that air pollution exposure is associated with a host of serious adverse human health effects. This documentation includes impacts revealed by observational epidemiology, and confirmed by controlled chamber exposures, showing consistent associations between air pollution and adverse impacts across a wide range of human health outcomes. As summarized in Figure 1, while exposures begin in the lungs, the adverse effects of air pollution can reach systemically throughout the human body.

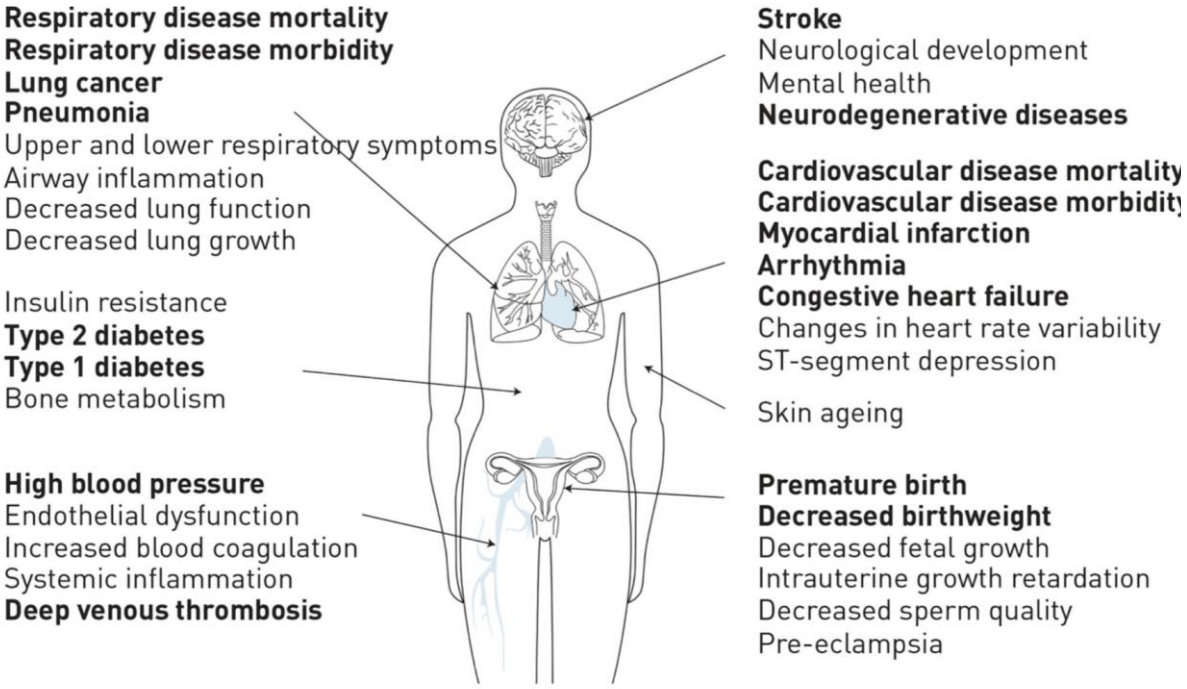


Figure 1. Overview of diseases, conditions and biomarkers affected by outdoor air pollution. Source: Thurston et al (2017)

While deaths from air pollution are the most severe outcome from air pollution exposures, Figure 2 below indicates that, for every death associated with air pollution, there is a pyramid of much greater numbers of morbidity effects, including hospital admissions, emergency department visits, doctor visits, missed work days, missed school days, asthma symptoms days, etc. Clearly, when the whole scope of other adverse health effects associated with these air pollution deaths is considered, there is no doubt as to the significance of these adverse health effects.

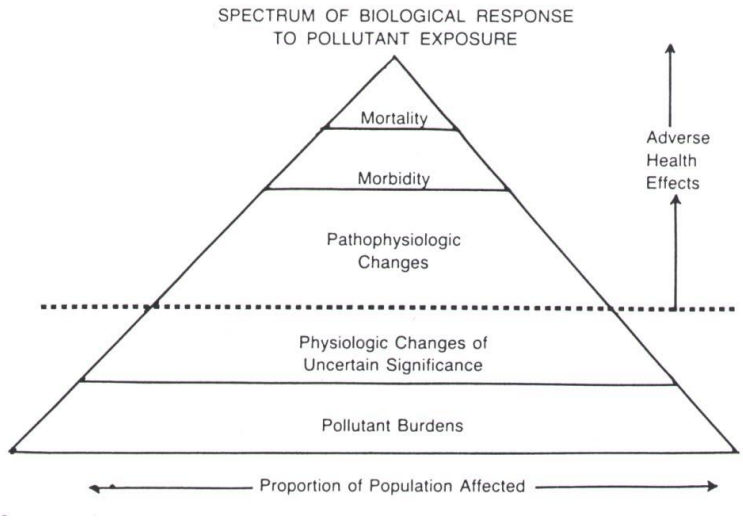


Figure 2. The Pyramid of Adverse Health Effects of Air Pollution on Health. (ATS, 1985)

Observational epidemiology studies provide the most compelling and consistent evidence of the adverse effects of air pollution. "Epidemiology" is literally "the study of epidemics," but includes all statistical investigations of human health and potentially causal factors of good or ill health. In the case of air pollution, such studies follow people as they undergo varying real-life exposures to pollution over time, or from one place to another, and then statistically inter-compare the health impacts that occur in these populations when higher (versus lower) exposures to pollution are experienced. In such studies, risks are often reported in terms of a Relative Risk

(RR) of illness, wherein a RR = 1.0 is an indication of no change in risk after exposure, while a RR>1.0 indicates an increase in health problems after pollution exposure. An RR significantly above 1 demonstrates that the air pollutant studied is damaging to health.

There are two types of epidemiological investigations: (a) population-based studies, in which an entire city's population might be considered in the analysis; and (b) cohort studies, in which selected individuals, such as a group of asthmatics, are considered. Both of these types of epidemiologic studies have shown confirmatory associations between air pollution exposures and increasing numbers of adverse impacts, including:

- decreased lung function (a measure of our ability to breathe freely);
- more frequent asthma symptoms;
- increased numbers of asthma and heart attacks;
- more frequent emergency department visits;
- increased incidence of new onset childhood asthma;
- additional hospital admissions; and
- increased numbers of deaths.

The fact that the adverse health effects of air pollution have been shown so consistently for so many health endpoints, in so many locales, and by so many different investigators using a variety of approaches, indicates these associations to be causal.

Ethnicity, age, and pre-existing medical conditions play a role in the extent of adverse health impacts from increased air pollution emissions. Analyses by me and by others in the field of air pollution health effects also indicate that underserved minorities and the poor are especially at risk from air pollution exposures (e.g., Gwynn and Thurston, 2001; Glad et al, 2012). Similarly, older adults are at greater risk of severe adverse outcomes from air pollution. Also, children, a

population known to be especially susceptible to the effects of air pollution because their bodies are developing (and because they spend larger amounts of time exercising outside) are an especially affected sub-population. This subpopulation of children can be expected to be among those most strongly affected by any increases in air pollution concentrations.

Exposures to nitrogen oxides (NOx) in the air have been associated with adverse human health effects, in addition to their being a precursor of (i.e., leading to the formation of) secondary PM2.5 and ozone in the atmosphere, which also have adverse health effects (US EPA, 2010). Nitrogen Dioxide (NO2) is one of a group of highly reactive gases containing both nitrogen and oxygen known as oxides of nitrogen or nitrogen oxides (NOx). NO2 primarily gets into the air we breathe from the combustion of fuels, including from diesel powered vehicles.

Short-term (acute) exposures to NO2, for as briefly as 1-hour or less in length, are known to aggravate existing respiratory diseases, particularly asthma, leading to episodes of respiratory symptoms (e.g., coughing, wheezing or difficulty breathing), hospital admissions, and/or visits to emergency rooms. Indeed, the U.S. EPA's Integrated Science Assessment (ISA) for Oxides of Nitrogen (EPA/600/R-15/068) has concluded that research studies have provided scientific evidence that is sufficient to infer a relationship to exist between short-term NO2 exposure and adverse effects on the respiratory system. These associations between ambient NO2 were found in a broad array of respiratory effects, ranging "from subclinical increases in pulmonary inflammation to respiratory mortality." The likely mechanistic pathways of such respiratory effects are summarized in Figure 3. The EPA ISA report concludes, and I agree, the scientific evidence shows that: "The NO2-induced increases in allergic inflammation and airway responsiveness in controlled human exposure studies of adults with asthma comprise the key evidence that NO2 exposure can independently exacerbate asthma and support the epidemiologic

evidence for asthma hospital admissions and ED visits, as well as symptoms, lung function decrements, and pulmonary inflammation in populations with asthma." (U.S. EPA, 2016).

Longer-term (e.g., annual average) exposures to elevated concentrations of NO2 contributes to the development of asthma, and can potentially increase susceptibility to respiratory infections. People with asthma, children, older adults, and those with pre-existing disease are generally at greater risk for the health effects of air pollutants like NO2.

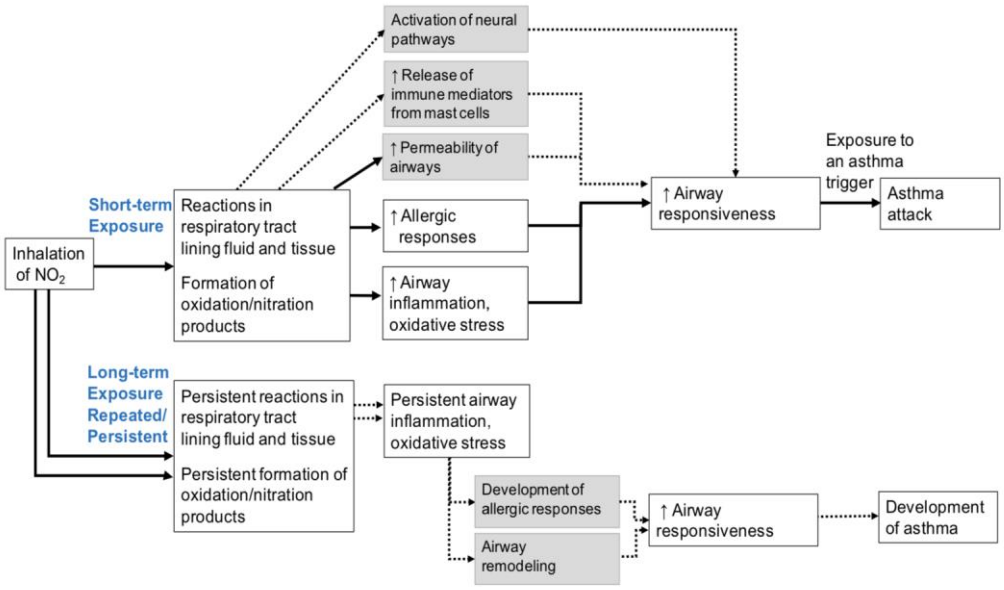


Figure 3. Biological Pathways of Nitrogen Dioxide (NO_2) Exposure Effects on the Lung (US EPA, 2016)

(Note: White boxes and solid arrows describe pathways well supported by available evidence, while grey boxes and dotted lines are for pathways for which evidence is less certain.)

One of the most severe health impacts associated with exposure to NO2 is the development of new onset childhood asthma. Traffic related air pollution (TRAP) exposures, including to NO2 air pollution, were evaluated as a cause of childhood or adult-onset asthma in the Health Effects Institute (HEI) Special Report 17 (2010). This publication concluded living near busy

roads was a risk factor for onset of childhood asthma, but data were insufficient to conclude causality at that time. However, several key studies on the topic have now been published since this report's release. For example, the Southern California Children's Health Study (CHS) found an increased risk of new-onset childhood asthma from TRAP at home residence (McConnell, 2010). Khreis and colleagues subsequently synthesized 41 studies that focused on children's TRAP exposures as a potential cause for asthma development, finding associations with TRAP metrics, especially NO2. (Figure 4). A 2017 meta-analysis of 18 studies of prenatal air pollution exposures and childhood asthma similarly found associations between risk of wheezing and asthma development in childhood with air pollution exposure, including NO_2 (Hehua, 2017).

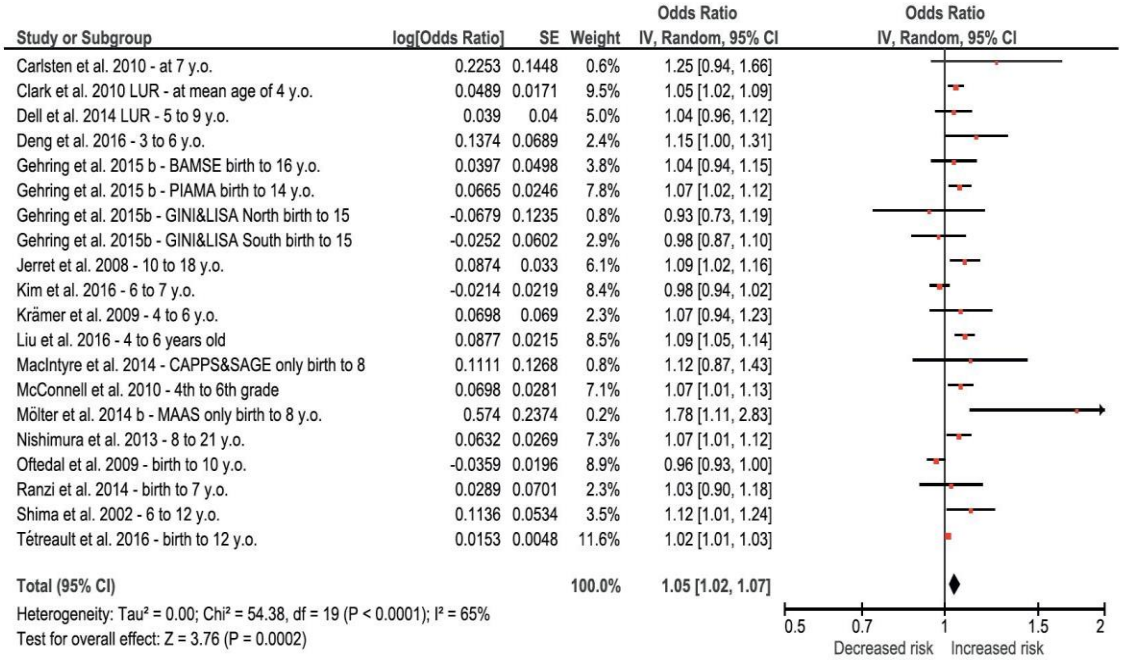
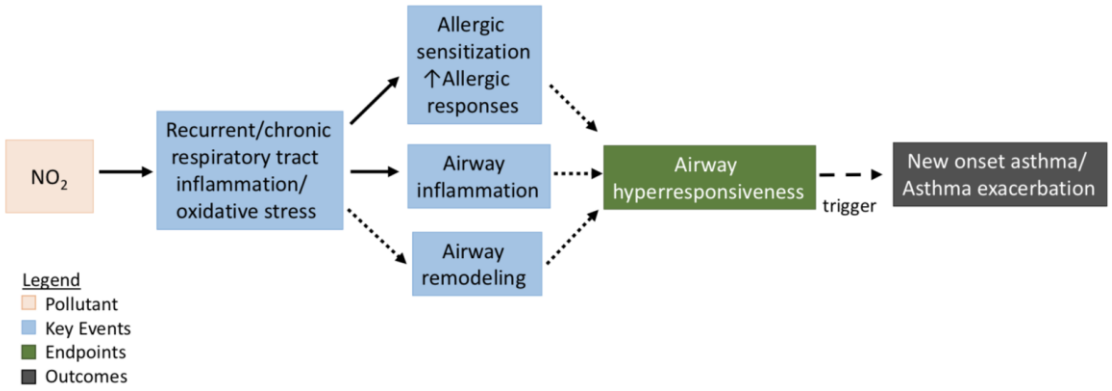


Figure 4. Meta-analysis of studies of NO_2 and new-onset asthma in children (Khreis, 2017)

Earlier this year, a well-designed multi-level longitudinal study drawn from three waves of CHS cohort recruitment during a decade of air pollution decline in Southern California found

that decreases in ambient NO_2 and $PM_{2.5}$ between 1993 and 2014 were significantly associated with lower asthma incidence (Garcia et al, 2019). This study is consistent with an inference of causality of the NO_2 air pollution-asthma incidence association, since an intervention to reduce exposure was followed by a reduction in disease incidence. A mechanistic biological pathway for the development of new onset asthma from NO_2 air pollution exposure is shown in Figure 5 (USEPA, 2016).



Note: Pathways indicated by a dotted line are those for which evidence is limited to findings from experimental animal studies, while evidence from controlled human exposure studies is available for pathways indicated by a solid line. The dashed line indicates a proposed link to the outcome of new onset asthma/asthma exacerbation. Key events are subclinical effects, endpoints are effects that are generally measured in the clinic, and outcomes are health effects at the organism level. NO_2 = nitrogen dioxide.

Source: National Center for Environmental Assessment.

Figure 5. Mechanistic biological pathway of childhood asthma development from NO_2 exposures (US EPA, 2016).

Using the available science, Achakulwisut and colleagues (2019) have estimated the annual global number of new pediatric asthma cases attributable to NO_2. They found that, globally, 4·0 million new pediatric asthma cases could be attributed to NO_2 pollution annually, accounting for 13% of global incidence.

Overall, the US EPA, in its most recent NO_2 ISA listed the health effects identified, including both quantified and non-quantified for its cost-benefits analyses, as shown in Table 1.

Table 1. Human Health Effects of NO_2 Air Pollution Identified by the US EPA (2010)

Pollutant / Effect	Quantified and Monetized in Primary Estimates [a]	Unquantified Effects [b, c] Changes in:
NO_2/Health	Asthma Hospital Admissions	Premature mortality
	Chronic Lung Disease Hospital Admissions	Pulmonary function
		Other respiratory emergency department visits
	Asthma ER visits	Other respiratory hospital admissions
	Asthma exacerbation	
	Acute Respiratory symptoms	

Based on the *above evidence, I conclude that exposures to NO_2 air pollution exposures are a significant contributor to adverse health effects, especially among the youngest of our society, and that the increased NOx emissions from VW TDI automobiles have resulted in increased adverse human health impacts.*

B. Increased NOx Emissions From VW TDI Automobiles Have Resulted In Increased Ozone, Which Has Resulted In Additional Adverse Human Health Impacts.

Ozone (O3) is a gaseous air pollutant that is not emitted directly by the diesel vehicles under consideration here, but is instead a secondary air pollutant formed in the atmosphere from pollutants emitted by these units, especially from nitrogen oxides and hydrocarbon emissions, which are both products of fossil fuel combustion. This is especially the case in the summer months, when there is more sunlight and warmer weather, which enhances the formation of O3 in the atmosphere

Ozone can irritate the human respiratory system when breathed, causing exposed people to cough, feel an irritation in the throat, and/or experience an uncomfortable sensation in the chest area. Ozone has also been shown to reduce the lung's ability to inhale and exhale, thereby making it more difficult for people to breathe as deeply and vigorously as they normally would (e.g., see Bates, 1995). Research shows that ozone can also acutely aggravate asthma, and new

evidence suggests that it may cause more children to get asthma. When ozone levels are high, people with asthma have more attacks that require a doctor's attention or the use of additional medication. One reason this happens is that ozone makes people more sensitive to allergens, which are the most common triggers for asthma attacks. Ozone can inflame and damage cells that line the human lung, and O3 has been compared by some to "getting a sunburn on your lungs." Ozone may also aggravate chronic lung diseases, such as emphysema and bronchitis, and can reduce the immune system's ability to fight off bacterial infections in the respiratory system.

Among the important adverse effects associated with ozone exposure to asthmatics is the triggering of asthma attacks. The effects of ozone air pollution on children with asthma have been demonstrated in my own research following a group of children at an asthma summer camp located in Connecticut. This study of a group of about 55 moderate to severely asthmatic children showed that these children experienced statistically significant reductions in lung function, increases in asthma symptoms, and increases in the use of unscheduled asthma medications as ozone pollution levels rose. As shown in Figure 6, the risk of a child having an asthma attack was found to be approximately 40 percent higher on the highest ozone days than on an average study day (Thurston et al., 1997). Consistent with other research in this area, there is no indication in this plot of a threshold concentration below which children with asthma are safe from the effects of ozone increases.

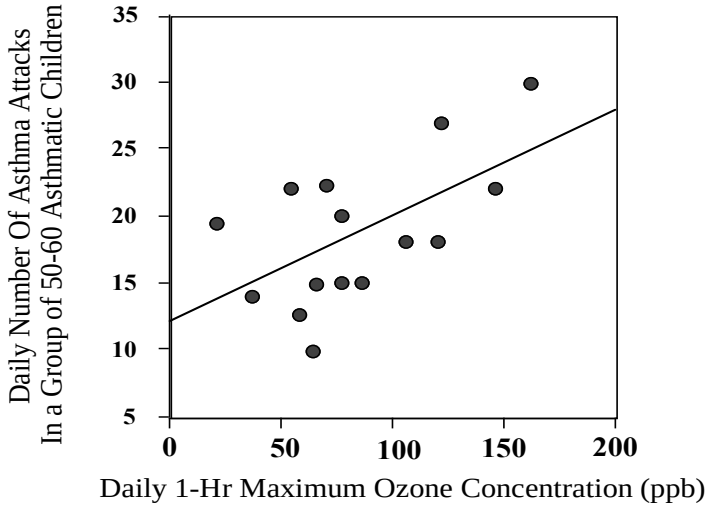


Figure 6. The number of asthma attacks among children at an "Asthma Camp" in Connecticut increase as the ozone levels rise (Source: Thurston *et al.*, 1997)

These asthma camp results were confirmed by a larger study published in the Journal of the American Medical Association (JAMA). Gent *et al.* (2003) presented a cohort study of asthmatic children from the New Haven, CT area, including 130 children who used maintenance medications for asthma and 141 children who did not. The more severe asthmatics were identified as those using maintenance medication. For these severe asthmatics, the study found that the level of O_3 exposure was significantly associated with worsening of symptoms and an increase in the use of rescue medication. Each 50 parts per billion (ppb) increase in 1-hour average O_3 was associated with an increased likelihood of wheezing (by 35%) and chest tightness (by 47%). The findings indicate that asthmatic children are particularly vulnerable to ozone, even at pollution levels below the prevailing U.S. EPA air quality standards.

My own research has also shown that ozone air pollution to be associated with diminished lung function in non-asthmatic healthy children at a YMCA summer camp in a pristine area in the Kittatinny Ridge, in the northwestern part of the state (Spektor *et al.*, 1988a). Similarly, in the summer of 1988, Berry *et al.* (1991) conducted a field health study at two summer day camps

in suburban-central New Jersey. Thirty-four campers and counselors had daily lung function tests, and it was found that the campers had a statistically significant decrease in peak expiratory flow rate associated with increasing ozone concentrations, indicating an acute loss in the children's ability to inhale and exhale after ozone exposure.

The adverse effects of exposure to ozone in ambient air on the lungs of individuals has been demonstrated in studies that I have conducted in the State of New York, as well. For example, respiratory function damage was demonstrated in a study I co-authored of 30 healthy adult non-smokers engaged in a regular daily program of outdoor exercise in Tuxedo, NY during the summer of 1985 (Spektor *et al.*, 1988b). All measured health indices showed statistically significant O_3-associated decreases in the lung function of the runners as ozone levels increased. More recently, using lung bronchoscopy (which allows a visualization of the main tubes of the lungs, by means of a flexible lighted instrument introduced through the vocal cords and windpipe) and broncho-alveolar lavage (BAL, or a washing of the lining of the lung), Kinney *et al.* (1996) examined some 19 normal volunteer joggers from Governors Island, NY. The joggers exercised in the afternoon during the 1992 summer season. These results indicate a significant inflammatory response in the lungs of recreational joggers in New York City exposed to regional ozone and associated co-pollutants during the summer months.

The O_3 - morbidity associations indicated by the above-presented epidemiological studies are also supported by a large body of data from controlled human exposure studies that give consistent and/or supportive results, and that have demonstrated pathways by which ozone can damage the human body when breathed. Clinical studies have demonstrated decreases in lung function, increased frequencies of respiratory symptoms, heightened airway hyper-responsiveness, and cellular and biochemical evidence of lung inflammation in healthy

exercising adults. For example, in controlled exposure studies, McDonnell *et al.* (1991) and Devlin *et al.* (1991) found that prolonged controlled exposures of exercising men to levels of ozone common in present-day U.S. (only 80 ppb) resulted in significant decrements in lung function, induction of respiratory symptoms, increases in nonspecific airway reactivity, and cellular and biochemical changes in the lung.

Ozone may also cause permanent lung damage. For example, repeated short-term ozone damage to children's developing lungs may lead to reduced lung function in adulthood (*e.g.*, *see* Kunzli et al, 1997). In adults, ozone exposure may accelerate the natural decline in lung function that occurs as part of the normal aging process (*e.g.*, *see* Detels, *et al.*, 1987). One important study suggests that long-term ozone exposure can increase the chances that children will develop asthma disease (McConnnell *et al.*, 2002).

Emergency Room Visits and Hospital Admissions are also increased by O_3 air pollution. Cody *et al.* (1992) analyzed data on New Jersey hospital emergency department (ED) visits for asthma, bronchitis, and finger wounds (a non-respiratory control) for the period May through August for 1988 and 1989, finding that, when temperature was controlled for in a multiple regression analysis, a highly significant relationship between asthma visits and ozone concentration was identified. In addition, a 5-year retrospective study by Weisel *et al.* (1995) of the association between ED visits for asthma with mean ambient ozone levels was conducted for hospitals located in central New Jersey. An association was identified in each of the years (1986-1990), and ED visits occurred 28% more frequently when the mean ozone levels were greater than 60 ppb O_3, as compared to when they were less than 60 ppb O_3.

Multi-city analyses have confirmed the ozone-mortality relationship. These include meta-analyses of multiple past ozone studies that show consistent associations between ozone and increases in mortality (Levy et al, 2005; Ito *et al.*, 2005; Bell *et al.*, 2005). In one analysis of some 95 U.S. cities over two decades published in JAMA, Bell et al (2004) showed that, even after controlling for particulate matter and weather, an increase of 10 parts-per-billion in daily ozone pollution was associated with approximately a 0.5% increase in daily risk of death. As discussed earlier, this size percent increase in daily admissions, though small, affects a huge portion of the population and accumulates day after day, week after week, and month after month, so that it accumulates to account for thousands of deaths each year in the U.S.

More recently, mortality effects from long-term exposure to ozone air pollution has now been confirmed in major cohort studies (Jerrett et al, 2009; Turner et al, 2016, Lim et al, 2019). In Jerrett et al, data from the study cohort of the American Cancer Society Cancer Prevention Study II were correlated with air-pollution data from 96 metropolitan statistical areas in the United States. 448,850 subjects, with 118,777 deaths in an 18-year follow-up period were considered. Data on daily maximum ozone concentrations were obtained from April 1 to September 30 for the years 1977 through 2000. Data on concentrations of fine particulate matter ($PM_{2.5}$) were obtained for the years 1999 and 2000. Associations between ozone concentrations and the risk of death were evaluated with the use of standard and multilevel Cox regression models. In single-pollutant models, ozone was associated with the risk of death from respiratory causes. The estimated relative risk of death from respiratory causes that was associated with an increment in ozone concentration of 10 ppb was 1.040 (95% confidence interval, 1.010 to 1.067). The association of ozone with the risk of death from respiratory causes was insensitive to adjustment for confounders and to the type of statistical model used. In a follow-up analysis of this same

database, Turner et al (2016) improved ozone exposure estimates by employing estimates of O_3 concentrations at the participant's residence, as derived from a hierarchical Bayesian space–time model. In two-pollutant models, adjusted for $PM_{2.5}$, significant positive associations remained between O_3 and all-cause (hazard ratio [HR] per 10 ppb, 1.02; 95% confidence interval [CI], 1.01–1.04), circulatory (HR, 1.03; 95% CI, 1.01–1.05), and respiratory mortality (HR, 1.12; 95% CI, 1.08–1.16) that were unchanged with further adjustment for NO_2.

Lim et al (2019) confirmed these ozone-mortality findings in another large cohort study conducted across the US. We investigated associations of long-term (annual or warm season average) O_3 exposure with all-cause and cause-specific mortality in the NIH-AARP Diet and Health Study, a large prospective cohort of U.S. adults with 17 years of follow-up from 1995 to 2011. Long-term annual average exposure to O_3 was significantly associated with deaths due to cardiovascular disease (per 10 ppb, HR=1.03; 95% CI: 1.01-1.06), ischemic heart disease (HR=1.06; 95% CI: 1.02-1.09), respiratory disease (HR=1.04; 95% CI: 1.00-1.09), and chronic obstructive pulmonary disease (HR=1.09; 95% CI: 1.03-1.15) in single-pollutant models.

Overall, there is a consistency between the epidemiologic study associations and experimental study results, supporting the conclusion that: 1) there is indeed a cause-effect relationship between the amount of air pollution and negative health effects; and, 2) there is no known threshold below which no effects are experienced. Thus, increases in air pollution from the excess nitrogen oxides emitted by the diesel vehicles in question resulted in commensurate increases in harm to public health from that air pollution, and the continued operation of these units continues those adverse health effects to accumulate.

Based on the above evidence, I conclude that exposures to ozone air pollution are a significant contributor to adverse health effects, and that the O_3 caused in the atmosphere by increased NOx emissions from VW TDI automobiles have resulted in increased adverse human health impacts.

C. Increased NOx Emissions From VW TDI Automobiles Have Resulted In Increased Particulate Matter, Which Has Resulted In Added Human Health Impacts.

In addition to lung damage, recent epidemiological and toxicological studies of $PM_{2.5}$ air pollution have shown adverse effects on the heart, including an increased risk of heart attacks. For example, when particulate matter stresses the lung (*e.g.*, by inducing edema), it places extra burden on the heart, which can induce fatal complications for persons with cardiac problems. Indeed, for example, Peters *et al.* (2001) found that elevated concentrations of fine particles in the air can elevate the risk of myocardial infarctions ("MIs") within a few hours, and extending up to one day after PM exposure. The Harvard University team found that a 48% increase in the risk of MI was associated with an increase of 25 *u*g/m^3 $PM_{2.5}$ during a two-hour period before the onset of MI, and a 69% increase in risk to be related to an increase of 20 *u*g/m^3 $PM_{2.5}$ in the twenty-four-hour average one day before the MI onset (Peters *et al.*, 2001). Numerous other U.S. studies have also shown qualitatively consistent acute cardiac effects, such as the Zanobetti and Schwartz (2006) study of hospital admissions through an emergency department for MI (ICD-9 code, and Zanobetti et al. (2009) that examined the relationship between daily PM2.5 concentrations and emergency hospital admissions for cardiovascular causes, MI, and congestive heart failure in twenty-six U.S. communities during 2000–2003.

Cardiac effects at the biological level have also been documented in both animal and human studies. Animal experiments at Harvard University by Godleski *et al.* (1996, 2000) indicate that

exposures to elevated concentrations of ambient particulate matter can result in cardiac-related problems in dogs that had been pre-treated (in order to try to simulate sensitive individuals) to induce coronary occlusion (i.e., narrowed arteries in the heart) before exposing them to air pollution. The most biologically and clinically significant finding was that, in these dogs, the PM affected one of the major electrocardiogram ("ECG") markers of heart attacks (myocardial ischemia) in humans, known as elevation of the ST segment.

Cardiac effects at the biological level have been found in human studies, as well. For example, Pope *et al.* (1999) and Gold *et al.* (2000) found that PM exposure is associated with changes in human heart rate variability ("HRV"). Such changes in heart rate variability may reflect changes in cardiac autonomic function and the risk of sudden cardiac death. In the Pope *et al.* study, repeated ambulatory ECG monitoring was conducted on seven subjects for a total of twenty-nine person-days before, during, and after episodes of elevated pollution. After controlling for differences across patients, elevated particulate levels were found to be associated with (1) increased mean heart rate; (2) decreased SDNN, a measure of overall HRV; (3) decreased SDANN, a measure that corresponds to ultra-low frequency variability; and (4) increased r-MSSD, a measure that corresponds to high-frequency variability. This confirms, at the individual level, that biological changes do occur in heart function as a result of PM exposure, supporting the biological plausibility of the epidemiological associations between PM exposure and cardiac illnesses.

Epidemiologic research conducted on U.S. residents has indicated that acute short-term exposures to PM air pollution are associated with increased risk of mortality. For example, a nationwide time-series statistical analysis of daily death counts by the Health Effects Institute (HEI, 2003) examined mortality and PM10 air pollution (a subset of particulate matter air

pollution that is less than 10 µm in diameter, including PM2.5) in ninety cities across the United States, finding that, for each increase of 10 µg/m3 in daily PM10 air pollution concentration, there is an associated increase of approximately 0.3% in the daily risk of death by the public. Indeed, and I concur, the most recent U.S. EPA Particulate Matter Integrated Science Assessment ("ISA") (USEPA, 2009) unequivocally states that "[t]ogether, the collective evidence from epidemiologic, controlled human exposure, and toxicological studies is sufficient to conclude that a causal relationship exists between short term exposures to PM2.5 and cardiovascular effects . . . and mortality."

My recent studies have found that long-term exposure to combustion-related fine particulate air pollution is an important environmental risk factor for cardiopulmonary and lung cancer mortality. In addition to the acute health effects associated with daily PM pollution, long-term exposure to fine PM is also associated with increased lifetime risk of death and has been estimated to take years from the life expectancy of people living in the most polluted cities, relative to those living in cleaner cities. For example, in the Six-Cities Study (that was a key basis for the setting of the original PM2.5 annual standard in 1997), Dockery et al. (1993) analyzed survival probabilities among 8,111 adults living in six cities in the central and eastern portions of the United States during the 1970s and 80s. The cities were: Portage, WI (P); Topeka, KS (T); a section of St. Louis, MO (L); Steubenville, OH (S); Watertown, MA (M); and Kingston-Harriman, TN (K). Air quality was averaged over the period of study in order to study long-term (chronic) effects. As shown in Figure 7, it was found that the long-term risk of death, relative to the cleanest city, increased with fine particle exposure, even after correcting for potentially confounding factors such as age, sex, race, smoking, etc.

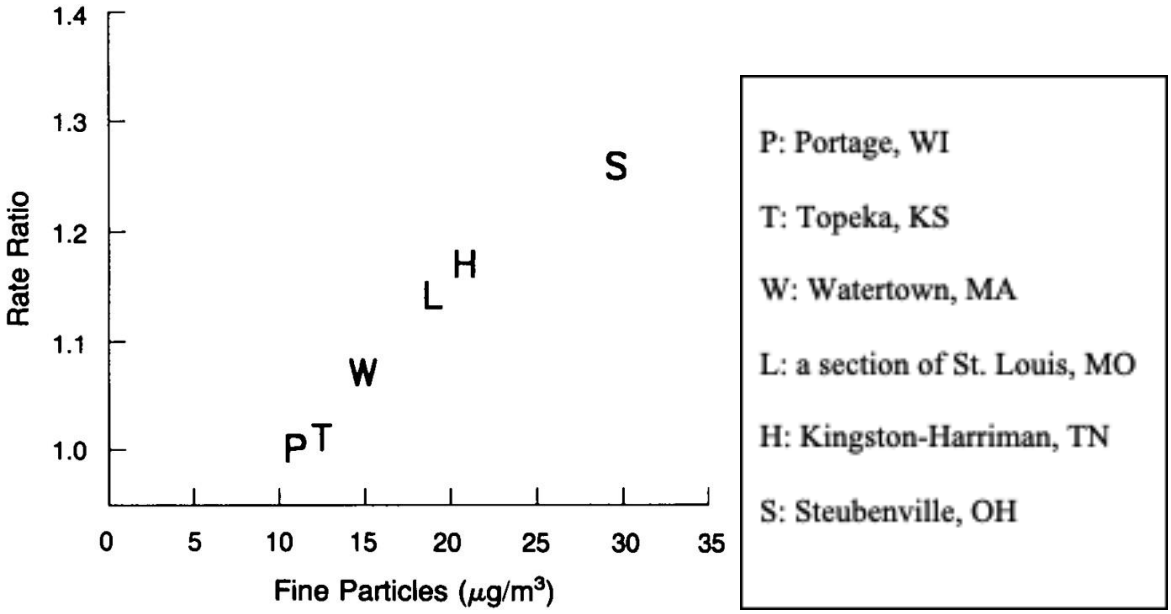


Figure 7. The Harvard Six-Cities Study showed that the lifetime risk of death increased across 6 U.S. cities as the average fine PM levels increased. (Source: Dockery et al., 1993.)

In addition, a study that I and co-authors published in the Journal of the American Medical Association ("JAMA"), shows that long-term exposure to combustion-related fine particulate air pollution is an important environmental risk factor for cardiopulmonary and lung cancer mortality. Indeed, as shown in Figure 8, this study indicates that the increase in risk of lung cancer from long-term exposure to $PM_{2.5}$ in a polluted city was of roughly the same size as the increase in lung cancer risk of a non-smoker who breathes passive smoke while living with a smoker, or about a 20% increase in lung cancer risk (*see* Pope, CA, *et al.*, 2002).

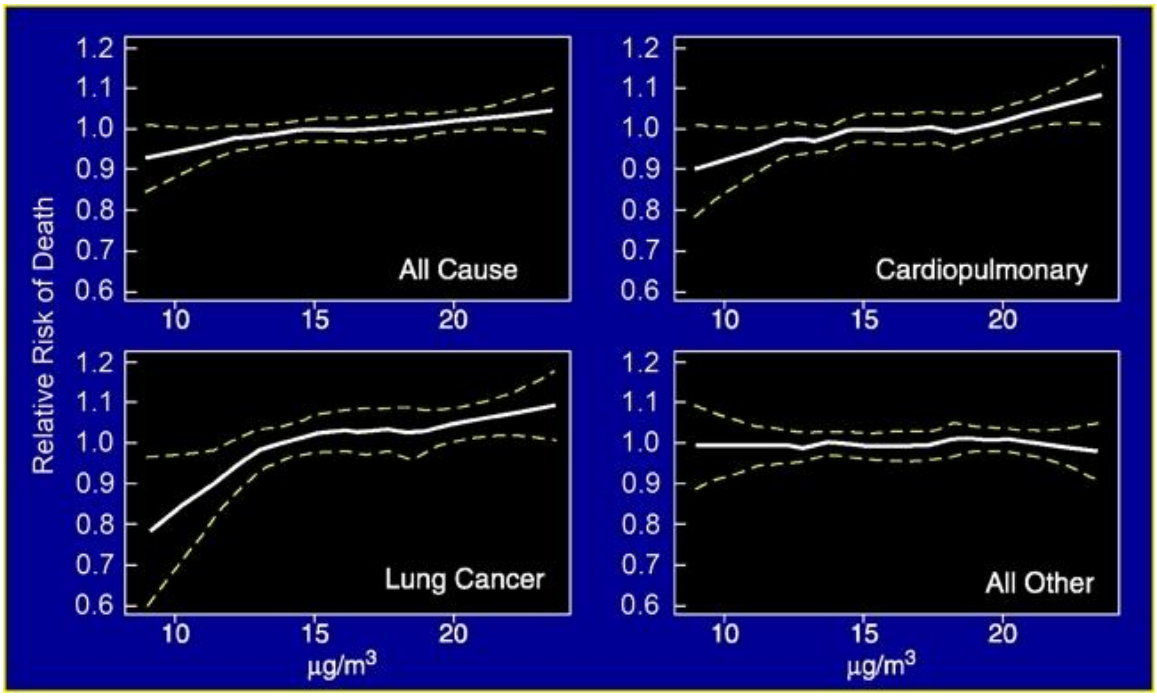


Figure 8. Cardiopulmonary and Lung Cancer Mortality Risks Increase Monotonically with Exposure to Long-Term Fine PM. (Adapted from: Pope, Burnett, Thun, Calle, Krewski, Ito, and Thurston, 2002.)

Moreover, long-term exposure to fine particles has been estimated to take more than a year from the life expectancy of people living in the most polluted cities, relative to those living in cleaner cities. For example, Brunekreef (1997) reviewed the available evidence of the mortality effects of long-term exposure to PM air pollution and, using life table methods, derived an estimate of the reduction in life expectancy implied by those effect estimates. Based on the results of Pope *et al.* (1995) and Dockery *et al.* (1993), a relative risk of 1.1 per 10 µg/m^3 exposure over fifteen years was assumed for the effect of fine PM air pollution on men 25–75 years of age. A 1992 life table for men in the Netherlands was developed for ten successive five-year categories that make up the 25–75 year old age range. Life expectancy of a twenty-five-year-old was then calculated for this base case and compared with the calculated life expectancy for the PM exposed case where the death rates were increased in each age group by a factor of 1.1. A difference of 1.11 years was found between the "exposed" and "clean air" cohorts'

overall life expectancy at age twenty-five. A similar calculation by the authors for the 1969–71 life table for U.S. white males yielded an even larger reduction of 1.31 years for the entire population's life expectancy at age twenty-five. Thus, these calculations indicate that differences in long-term exposure to ambient $PM_{2.5}$ can have substantial effects on life expectancy.

In my own research, I have found that acute (short-term) increases in PM air pollution are associated with increases in the number of daily asthma attacks, hospital admissions, and mortality. In particular, I have found that particulate matter air pollution is associated with increased numbers of respiratory hospital admissions in New York City; Buffalo, NY; and Toronto, Ontario, as well as with mortality in cities such as Chicago, IL; and Los Angeles, CA (*see, e.g.*, Thurston *et al.*, 1992). My results have been confirmed by other researchers considering locales elsewhere in the U.S. and throughout the world (*see, e.g.*, Schwartz, J., 1997).

With regard to long-term $PM_{2.5}$ exposures, I was a Principal Investigator of the above discussed ACS study published in the Journal of the American Medical Association (JAMA) in March of 2002, that shows that long-term exposure to combustion-related fine particulate air pollution is an important environmental risk factor for cardiopulmonary and lung cancer mortality (Pope *et al.*, 2002). More recently, my research has verified (as shown in Figure 5) that the association between $PM_{2.5}$ air pollution and cardiovascular mortality extends down to very low $PM_{2.5}$ concentration levels in the US as well (Thurston *et al*, 2016).

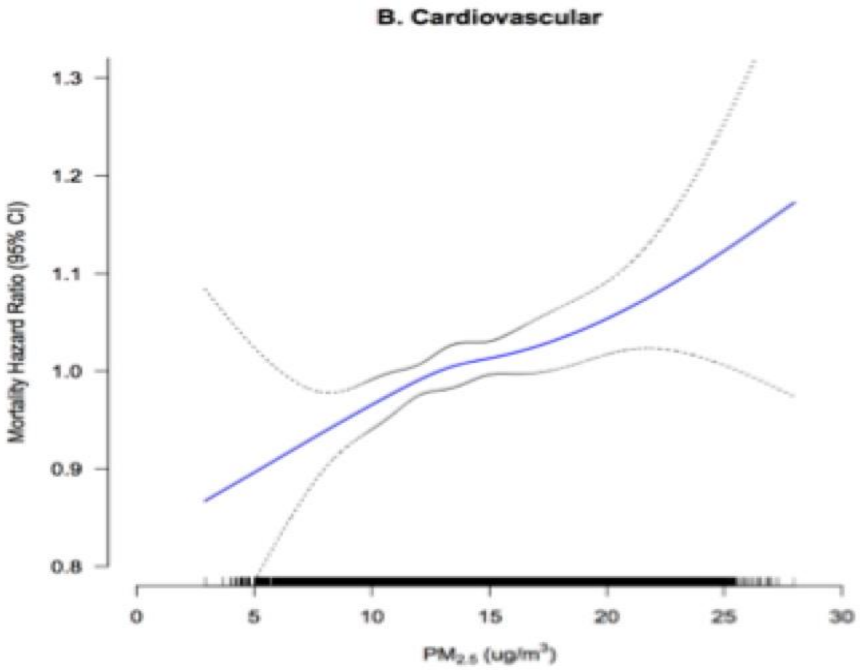


Figure 9. Mortality Risk from Cardiovascular Disease Increases with Rising $PM_{2.5}$ Exposure, Even Well Below the Present US Ambient Air Quality Standard annual limit for PM2.5 (12 µg/m^3). (Source: Thurston *et al*, 2016).

Importantly, this study is highly regarded, as it was conducted in a well characterized and large US population: the National Institutes of Health – American Association of Retired Persons (NIH-AARP) Diet and Health Study cohort. The NIH-AARP Study was initiated when members of the AARP, aged 50 to 71 years from 6 US states (California, Florida, Louisiana, New Jersey, North Carolina, and Pennsylvania) and 2 metropolitan areas (Atlanta, Georgia, and Detroit, Michigan), responded to a mailed questionnaire in 1995 and 1996. The NIH-AARP cohort questionnaires elicited information on demographic and anthropometric characteristics, dietary intake, and numerous health-related variables (*e.g.*, marital status, body mass index, education, race, smoking status, physical activity, and alcohol consumption), that was used to control for these factors in the air pollution mortality impact assessment. An extended analysis of the $PM_{2.5}$

- cardiovascular mortality association in the NIH-AARP Cohort has shown statistically significantly increased CVD mortality effects in subjects exposed to 8 to 12 µg/m^3 of long-term average $PM_{2.5}$ vs. those participants who resided in areas with concentrations less than 8 µg/m^3 (Hayes et al, 2019). This even more definitively confirms that the effects of $PM_{2.5}$ occur at levels below the prevailing ambient air quality standard (12 µg/m^3), and is consistent with the conclusion that $PM_{2.5}$ is a non-threshold air pollutant, meaning it can have severe adverse health impacts at any level of exposure.

Based on *the above evidence, I conclude that exposures to particulate matter air pollution are a significant contributor to adverse health effects, and that the $PM_{2.5}$ caused in the atmosphere by increased NOx emissions from VW TDI automobiles have resulted in increased adverse human health impacts.*

Date: December 2, 2019

George D. Thurston, SC. D

5. APPENDICES

A. List of Documents Considered In Forming Opinion:

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C. List of Cases In Which Expert Has Given Testimony At Trial Or In Deposition In The Last 4 Years:

Katie Lowery, et al., v. Honeywell International Inc., et al., (CV-2005-1749)